species-distribution by means of a defective replication system,

wherein the defective replication system has a rate of misincorporation for nucleotides higher than a rate of misincorporation of the viral wild-type replication system and,

C3 cont'd

wherein the viruses are replicated by the replication system having the higher rate of misincorporation at least as effectively as it is done by the replication system of the wild-type virus.

B1 cont'd

48. A process according to claim 47,

wherein a replication of a consensus-sequence comprising a nucleic acid sequence of the wild-type virus, is affected negatively in relation to other replicatable nucleic acids.

Sub C3

49. A process according to claim 47,

wherein the defective replication of the viral nucleic acid is induced by reaction of a chemical substance.

50. A process according to claim 49,

wherein the chemical substance is selected from the group consisting of an antimetabolite and an allosteric effector of the replication system.

51. A process according to claim 47,

wherein the defective replication is selected from the group consisting of a variant of a natural mutant spectrum of the quasi-species and a mutant produced by mutagenesis.

C3 cont.

52. A process according to claim 47,

wherein via the infiltration of a viral replication system into the virus population with subsequent infection of the target cells of the virus infection or by direct infiltration of a viral replication system or components of a viral replication system into the target cells, the target cells are enabled to replicate an infectious wild-type virus above the replication error threshold of the viral replication system, and to replicate with higher replication error rate than those of the respective stable quasi-species-distribution, having at least the same efficiency of replication.

B1 cont'd

53. A process according to claim 47,

wherein the replication systems RNA or DNA are selected from the group consisting of polymerases and co-factors of RNA or DNA polymerases.

54. A process according to claim 47,

wherein the infiltration of the defective replication system into the virus population occurs by transformation of individuals of the respective virus population or of the target cell in a manner according to the gene therapy.

C3 cont.

55. A process according to claim 47,

wherein the infiltration of the defective replication system occurs by superinfection of the target cell with defective viruses of the same species which carry the defective replication system.

B1 cont'd

56. A process according to claim 47,

wherein the gene carrying the viral replication system with the higher replication error rate was obtained by clonal selection or was synthetically prepared, and was infiltrated into a virus individual or into a target cell by a genetechnical procedure.

57. A process according to claim 47,

wherein the gene coding for the viral replication system with the higher error rate is provided with further regulatory gene

segments which take over further control functions in the transformed virus individual or in the transformed target cell.

58. A process according to claim 57,

wherein the further regulatory gene segment takes care for a higher replication rate of the virus population.

59. A process according to claim 48,

wherein the other replicatable nucleic acid is more effectively replicated than the nucleic acid of the consensus-sequence.

60. A process according to claim 48,

wherein a characteristic superiority parameter s is diminished by a combination of the replication system and one or more nucleases and/or ribozymes and/or antisense-RNA, whereby one or more nucleases and/or ribozymes and/or antisense-RNA are directed to components of the respective virus genome and/or the other replicatable nucleic acid is present in the not infected target cell only in a minor concentration in the form of replicator or replicator precursor, and will be replicated only after the infection by the polymerase of the infected virus.

61. A process for the treatment or prophylaxis of viral diseases, whereby either the affected target cells are transformed with a vector system, or the target cells are transformed by infiltration of a viral system which is leading to a higher error rate of rate of misincorporation, or the target cells are treated with one or more substances which cause an increased rate of misincorporation of the replication.

C3 cancel

62. A process according to claim 47,

B1 cont'd

wherein host cells are the target cells of the viral infection and are selected from the group consisting of monocellular organisms, bacteria, plant cells, animal host cells, blood cells, and erythropoietic stem cells.

63. An agent for the performance of the process according to claim 47, comprising a nucleic acid or a nucleic acid coding for a nucleic acid obtained by reaction of nucleotides and a viral replication system as well as other factors which are necessary for the reproduction of viruses under formation of oligo- or polynucleotides, whereby it is exclusively selectioned towards maximum amplification of the oligo- or polynucleotides by the viral replication system.

64. An agent according to claim 63, comprising at least one gene segment coding for a viral replication system and/or a co-factor of a viral replication system,

wherein the system to be coded is leading to a viral replication system with a higher rate of misincorporation than fixed by the native replication system, whereby the efficiency of the replication is at least maintained.

65. An agent according to claim 63, comprising together with the replication system, which is leading to higher rates of misincorporation, transformed viruses, phages or eucaryotic cells or procaryotic cells and/or respectively prepared phages or plasmids for the transformation of the target cell or transformed target cells themselves.

66. An agent according to claim 63,

wherein they are replication enzymes and cause a replication above the inherent error threshold under an at least equal replication efficiency as compared with the wild-type.

67. A method of destabilizing viral quasi-species distributions without inducing resistance to therapeutic agents

comprising inducing defective replication of nucleic acids of the viruses present in the quasi-species distribution around a consensus sequence by

replicating the nucleic acids by a defective replication system that has a rate of nucleotide misincorporation higher than the rate of nucleotide misincorporation of the viral wild-type replication system and a replication efficiency at least as great as the wild-type replication system.

B1 cont'd

68. The method according to claim 67,

wherein the defective replication system results from a natural mutation of the quasi-species distribution or is produced by mutagenesis.

69. A method for the treatment or prophylaxis of diseases caused by a virus comprising inducing a rate of misincorporation during viral replication higher than the rate of misincorporation of the wild-type virus by

a) transforming target cells with a vector system having at least one viral replication system having a rate of nucleotide misincorporation higher than the wild type

viral replication system,

b) transforming the target cells by introduction of a viral system with a higher rate of nucleotide misincorporation, or

c) treating target cells with one or more substances that cause an increased rate of nucleotide misincorporation of viral replication.

B'

(Sub C3)

70. The method according to claim 67,

wherein the viral quasi-species distribution is further destabilized by one or more nucleases, ribozymes, antisense-RNA, or combinations thereof directed to components of the virus.

71. The method according to claim 67,

wherein the destabilization of viral quasi-species distribution occurs in plant cells or animal cells.

(Sub C3)

72. A process according to claim 61,

wherein the affected target cells are transformed with a vector system comprising a viral vector system, having at least one